Probiotic Use in Gastrointestinal (G.I.T.) Infections

Dr. Pradyuman Kaushik [1*], Dr. J Srinivas [2], Dr. Rati Saxena [3], Dr. Prashant Jr [4]
[1-3] Department of Microbiology, Raj Shree Medical College, Bareilly, Uttar Pradesh, India
[4] Dr. KNS Memorial Institute of Medical Sciences, Barabanki, Uttar Pradesh, India

* Corresponding Author: **Dr. Pradyuman Kaushik**

Article Info

ISSN (online): 2582-8940
Volume: 07
Issue: 03
Received: 11-05-2026
Accepted: 09-06-2026
Published: 07-07-2026
Page No: 61-65

Abstract
Probiotics — live microorganisms that, when administered in adequate amounts, confer a health benefit on the host — have been widely investigated as adjunctive or stand-alone therapy for gastrointestinal (GIT) infections. This narrative review synthesises evidence from systematic reviews, meta-analyses, large randomised controlled trials, and clinical guidelines on probiotic use in acute infectious diarrhoea, antibiotic-associated diarrhoea (AAD), Clostridioides difficile-associated diarrhoea (CDAD), Helicobacter pylori infection, and traveller's diarrhoea. Relevant literature was identified through PubMed, the Cochrane Library, and Google Scholar. Pooled evidence indicates that selected strains — particularly Lactobacillus rhamnosus GG and Saccharomyces boulardii — can reduce the duration and severity of acute diarrhoea and lower the incidence of AAD and CDAD, while multi-strain formulations modestly improve H. pylori eradication rates and reduce therapy-related adverse effects. However, an updated 2020 Cochrane review and two large paediatric emergency-department trials have tempered earlier optimism, underscoring strain specificity, dosing, timing, and publication bias as major sources of heterogeneity. Probiotic supplementation appears safe in immunocompetent populations and offers measurable, though strain- and indication-dependent, benefit, supporting cautious, evidence-guided clinical use.

DOI: https://doi.org/10.54660/IJMBHR.2026.7.3.61-65

Keywords: Probiotics, Gastrointestinal infections, Diarrhoea, Lactobacillus rhamnosus GG, Saccharomyces boulardii, Clostridioides difficile

1. Introduction

Gastrointestinal (GIT) infections — including acute infectious diarrhoea, antibiotic-associated diarrhoea (AAD), Helicobacter pylori-related disease, and Clostridioides difficile-associated diarrhoea (CDAD) — remain a major cause of global morbidity and healthcare utilisation, particularly among children and hospitalised adults. Management relies on rehydration, supportive care, and antimicrobial therapy where indicated; however, rising antimicrobial resistance and antibiotic-associated dysbiosis have intensified interest in adjunctive, non-antibiotic strategies.

Probiotics are defined by the International Scientific Association for Probiotics and Prebiotics (ISAPP), building on the FAO/WHO definition, as live microorganisms that confer a health benefit on the host when administered in adequate amounts [1]. Commonly studied genera include *Lactobacillus*, *Bifidobacterium*, and the yeast *Saccharomyces boulardii*, acting through competitive exclusion, antimicrobial metabolite production, barrier enhancement, and immunomodulation [14,15] (Figure 1).

A substantial body of randomised controlled trials, systematic reviews and meta-analyses has examined probiotics across acute infectious diarrhoea, AAD, CDAD prevention, adjunctive *H. pylori* eradication, and traveller's diarrhoea (TD) prevention, but

conclusions have not been uniform; some earlier positive findings have been tempered by larger trials and updated reviews accounting for publication bias [2,3,8,9]. This review synthesises current evidence on probiotic use in gastrointestinal infections, summarising the strains and indications with the strongest data, the magnitude of reported effects, underlying mechanisms, and clinical caveats.

2. Methodology

A narrative review was conducted using PubMed, the Cochrane Library, and Google Scholar for publications from January 2003 to mid-2026, combining "probiotics" with "gastrointestinal infection," "acute diarrhoea," "antibiotic-associated diarrhoea," "Clostridioides difficile," "Helicobacter pylori," and "traveller's diarrhoea." Priority was given to Cochrane systematic reviews, meta-analyses, large multicentre RCTs, and guidelines from recognised gastroenterology/paediatric societies [2-13].

Inclusion criteria: human studies, English-language, a defined GIT infectious indication (acute infectious diarrhoea, AAD, CDAD, H. pylori infection, or TD), and a quantifiable outcome (duration, incidence, eradication rate, or relative/odds risk). *In vitro* and animal studies, case reports, and non-infectious indications (e.g., irritable bowel syndrome, inflammatory bowel disease) were excluded as outside the stated scope.

For each source, the probiotic strain(s), GIT indication, effect estimate (with 95% CI where available), and proposed mechanism were extracted and tabulated (Tables 1-3, Figures 1-2). Given the narrative, rather than formal PRISMA-based systematic, design, findings were synthesised qualitatively, with quantitative estimates drawn directly from the cited meta-analyses for illustrative comparison.

3. Results

Table 1 summarises the probiotic strains/formulations most frequently studied across the five GIT infection contexts identified, with their reported clinical effects.

Table 1: Major probiotic strains/formulations studied in gastrointestinal infections and their primary clinical indications

Strain / formulation	Primary GIT indication	Reported clinical effect	Ref.
Lactobacillus rhamnosus GG	Acute (largely viral) paediatric gastroenteritis	Earlier meta-analyses: ~1-day shorter diarrhoea; large 2018 ED trial found no benefit	7, 8
Saccharomyces boulardii	Antibiotic-associated diarrhoea; adjunct in CDAD/recurrent CDI	Significant reduction in AAD incidence (21 RCTs); only agent with evidence for recurrent CDI	5, 6
Multi-strain Lactobacillus mixtures	CDAD / AAD prevention during antibiotic exposure	Pooled RR reduction in CDAD and AAD incidence vs placebo	4, 5
Multi-strain Lactobacillus + Bifidobacterium	Adjunct to H. pylori eradication therapy	Modest improvement in eradication rate; consistent reduction in antibiotic-related side effects	12
S. boulardii / Lactobacillus-Bifidobacterium combinations	Prevention of traveller's diarrhoea (TD)	Significant overall reduction in TD incidence; smallest effect size of all indications reviewed	13

Acute infectious diarrhea: The largest evidence base concerns acute infectious (largely viral) diarrhoea, mainly in children. The original Cochrane review by Allen *et al*. [2] (63 RCTs, >8,000 participants) found probiotics reduced the risk of diarrhoea persisting ≥3 days (RR 0.66, 95% CI 0.55-0.77) and shortened mean duration by roughly one day (Table 2, Figure 2). However, the 2020 update by Collinson *et al*. [3] (82 RCTs, 12,127 participants), after identifying substantial publication bias among smaller trials, concluded the overall evidence was no longer clinically convincing under GRADE assessment (Table 2).

Antibiotic-associated and C. difficile-associated diarrhea: For AAD, McFarland's meta-analysis [5] reported a pooled RR ≈ 0.43 favouring probiotics, with *Lactobacillus* combinations and *Saccharomyces boulardii* the most consistently effective. *S. boulardii* specifically reduced AAD incidence across 21 RCTs with no excess adverse events [6]. For CDAD prevention, Goldenberg *et al*. [4] (23 RCTs, 4,213 participants) found probiotics safe and effective, with the largest relative-risk reduction examined here (RR ≈ 0.36, 95% CI 0.26-0.51) (Table 2, Figure 2).

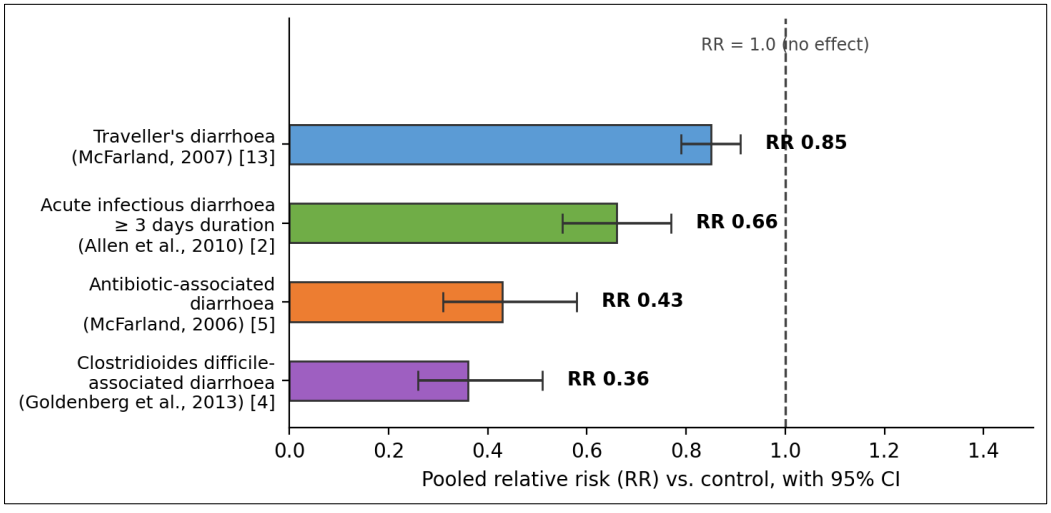

Fig 1: Pooled relative-risk estimates for probiotic supplementation across selected gastrointestinal infection outcomes (RR < 1.0 favours probiotics; 95% CI shown)

Table 2: Pooled effect estimates from key systematic reviews and meta-analyses of probiotic use in GIT infections

Outcome	Source review	Trials / N	Pooled estimate (95% CI)	Direction
Acute infectious diarrhoea (≥3 days)	Allen *et al*., 2010	63 RCTs; ~8,014	RR 0.66 (0.55–0.77)	Reduced risk
Acute infectious diarrhoea (update)	Collinson *et al*., 2020	82 RCTs; 12,127	No convincing effect (publication bias; GRADE)	Uncertain
Antibiotic-associated diarrhoea	McFarland, 2006	Pooled RCTs	RR ≈ 0.43 (0.31–0.58)	Reduced risk
C. difficile-associated diarrhoea	Goldenberg *et al*., 2013	23 RCTs; 4,213	RR ≈ 0.36 (0.26–0.51)	Reduced risk
Traveller's diarrhoea prevention	McFarland, 2007	12 treatment arms	RR 0.85 (0.79–0.91)	Reduced risk
Paediatric acute gastroenteritis (ED)	Schnadower/Freedman, 2018	943 + 886 children	No significant reduction in moderate-severe AGE	No effect

Helicobacter pylori eradication: Adjunctive probiotics during standard triple/quadruple eradication therapy modestly improve eradication rates and, more consistently, reduce therapy-related adverse effects such as diarrhoea, nausea and vomiting [12]. Multi-strain *Lactobacillus*/*Bifidobacterium* combinations given for >2 weeks, before or alongside antibiotics, appear most beneficial (Table 1).

Traveller's diarrhea: McFarland's meta-analysis of 12 treatment arms [13] found probiotics significantly reduced TD risk (RR 0.85, 95% CI 0.79-0.91) — the smallest relative effect summarised here — with *S. boulardii* and specific *Lactobacillus*/*Bifidobacterium* combinations performing best (Table 1, Figure 2).

Paediatric trials and mechanisms: Two large 2018 multicentre placebo-controlled ED trials — Schnadower *et al*. [8] (*L. rhamnosus* GG, 943 children) and Freedman *et al*. [9] (*L. rhamnosus*/*L. helveticus*, 886 children) — found no significant reduction in moderate-to-severe gastroenteritis, despite earlier positive meta-analyses [7] (Table 2). The mechanisms underlying the effects above — competitive exclusion, antimicrobial production, toxin neutralisation, barrier enhancement, and immunomodulation [14,15] — are summarised in Figure 1 and Table 3, with their relative contribution varying by strain and infection context.

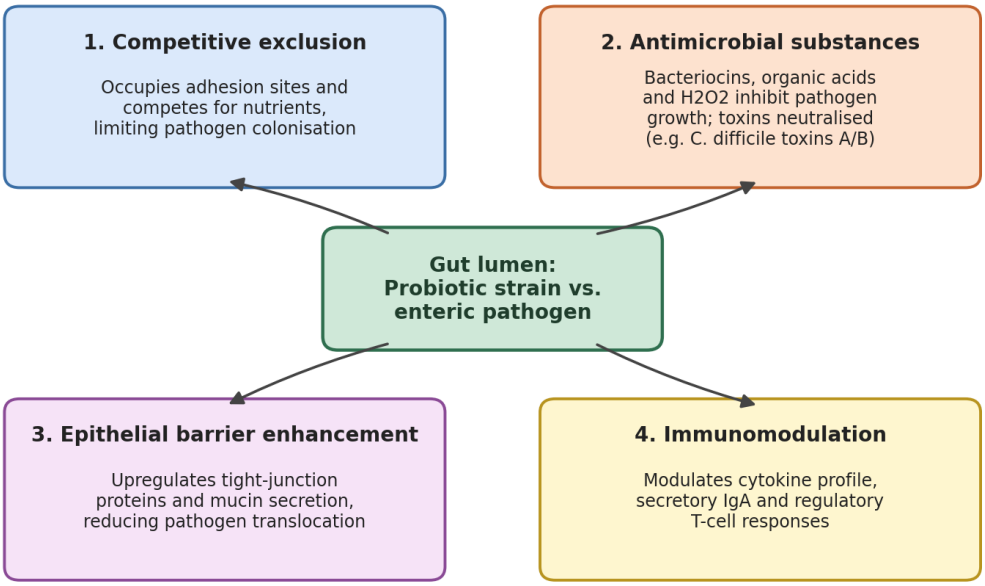

Fig 2: Schematic overview of proposed mechanisms by which probiotics counter enteric pathogens in gastrointestinal infections

Table 3: Reported mechanisms of action of probiotics relevant to gastrointestinal infections

Mechanism	Description	Example strains
Competitive exclusion	Competes for mucosal adhesion sites and nutrients, limiting pathogen colonisation	Lactobacillus, Bifidobacterium spp.
Antimicrobial production	Bacteriocins, organic acids and H2O2 inhibit growth of pathogenic organisms	L. casei, multi-strain mixtures
Toxin neutralisation	Degrades/neutralises bacterial toxins, e.g. C. difficile toxins A and B	Saccharomyces boulardii
Barrier enhancement	Upregulates tight-junction proteins and mucin secretion, reducing pathogen translocation	Multi-strain formulations
Immunomodulation	Modulates cytokine profile, secretory IgA and regulatory T-cell responses	Lactobacillus rhamnosus GG

4. Discussion

Probiotic efficacy in GIT infections is strain-, dose- and indication-specific rather than uniform. The largest relative-risk reductions were for CDAD (RR ≈ 0.36) [4] and AAD (RR ≈ 0.43) [5] prevention — contexts where the probiotic's task (limiting antibiotic-induced dysbiosis) aligns with competitive exclusion, antimicrobial metabolite production and toxin neutralisation, particularly by *S. boulardii* [6,15] (Figure 1). The smaller effect for TD (RR 0.85) [13] reflects the more heterogeneous, less predictable pathogen exposure during travel.

The evidence trajectory for acute infectious diarrhoea is instructive: earlier reviews [2] suggested moderate benefit, but the 2020 Cochrane update [3], incorporating large low-risk-of-bias trials and formally assessing publication bias, substantially weakened this conclusion — reinforced by two large 2018 paediatric ED RCTs [8,9] that found no benefit for *L. rhamnosus* GG or *L. rhamnosus*/*L. helveticus* combinations despite earlier positive meta-analyses [7]. This illustrates how pooled small-trial estimates can overstate effects not replicated in adequately powered studies.

Clinically, ESPGHAN/ESPID [10] and AGA [11] guidelines reflect this nuance, avoiding blanket recommendations for acute paediatric gastroenteritis while supporting strain-specific use (e.g., *S. boulardii* or *Lactobacillus* mixtures for AAD/CDAD prevention) where evidence is stronger. For *H. pylori*, probiotics are best framed as an adjunct reducing side effects and supporting antibiotic adherence rather than a substitute for standard regimens [12].

Mechanistically (Figure 1, Table 3), this variability reflects strain specificity: toxin neutralisation and competitive exclusion are well-documented for specific organisms (e.g., *S. boulardii* against *C. difficile* toxins [15]) but cannot be generalised across all “probiotic” products, which differ in species, strain, dose and viability. Safety data were reassuring for immunocompetent hosts, with no significant excess adverse events [4,6], though probiotic-associated bacteraemia/fungaemia in severely immunocompromised patients warrants caution [11].

Limitations include this review's narrative rather than systematic design, reliance on published pooled estimates rather than individual-patient data, and cross-study heterogeneity in strains, doses and formulations, which limits direct comparability of the Figure 2 estimates.

5. Conclusion

Probiotic supplementation has a defensible, evidence-based role in selected gastrointestinal infections — most notably prevention of antibiotic-associated and *Clostridioides difficile*-associated diarrhoea, and as an adjunct reducing side-effect burden during *Helicobacter pylori* eradication. Evidence for acute infectious and traveller's diarrhoea is more modest and increasingly nuanced following recent large trials. Recommendations should be strain- and indication-specific rather than generic, favouring formulations (e.g., *Saccharomyces boulardii*, specific *Lactobacillus* combinations) with the strongest evidence for the intended indication, with future research prioritising adequately powered, strain-specific trials with standardised outcomes.

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How to Cite This Article
Kaushik P, Srinivas J, Saxena R, Jr P. Probiotic use in gastrointestinal (G.I.T.) infections. International Journal of Medical and All Body Health Research. 2026;7(3):61-65. doi:10.54660/IJMBHR.2026.7.3.61-65.